Lead Poisoning From Retained Bullets

Pathogenesis, Diagnosis, and Management

MARC A. LINDEN, M.D.,* WILLIAM I. MANTON, PH.D.,† R. MALCOLM STEWART, M.D.,* ERWIN R. THAL, M.D.,‡ HOWARD FEIT, M.D., PH.D.*

Lead intoxication (plumbism) from retained bullets has rarely been reported but may be fatal if unrecognized. Bullets lodged within joint spaces or pseudocysts are more likely to develop this complication, although patients with retained missiles in other locations may also be at risk. Subtle findings such as the occurrence of unexplained anemia, abdominal colic, nephropathy, or neurologic deterioration in patients with retained missiles may suggest consideration of plumbism. An intercurrent metabolic stress such as infection, endocrinopathy, or alcoholism may be a precipitating factor. Among the various diagnostic studies available, mass spectrometric stable isotope dilution analysis may be the most reliable. It is important to employ chelation therapy prior to any operative intervention. This will reduce the mobilization of lead from bone during or following the surgical procedure.

LEAD INTOXICATION (plumbism) from retained lead missiles (bullets, shrapnel, buckshot) has been infrequently reported. A review of the world literature reveals 13 cases (Table 1) of lead poisoning from retained projectiles in which the diagnosis was documented by elevated levels of lead in either blood or urine. Three additional cases of lead poisoning from gunshot wounds have been diagnosed at this institution over the past year. This form of lead poisoning may be more common than previously considered. The protean manifestations of plumbism, a low index of suspicion, and the problems with accurate measurement of lead add to the difficulty in making a diagnosis. The availability of definitive therapy, however, calls for a heightened awareness of this entity.

Case Reports

Case 1. A 54-year-old black woman was admitted for evaluation of anemia. Five months previously she had sustained a gunshot wound to the right leg, with multiple bullet fragments lying adjacent to the distal femur, and possibly within the synovial fluid of the suprapatellar bursa. The patient had no occupational, environmental, or other exposure to lead. Three months prior to admission, she noted onset of midepigastric pain, which was aching in quality, nonradiating, and unrelated to food ingestion. Radiologic examination of the stomach, upper and lower intestines, and gall bladder, as well as proctoscopy, were normal. Physical examination revealed longstanding arthritic changes but no other specific abnormalities. No knee effusions were present, and the neurologic examination was within normal limits. No occult blood was found in fecal specimens. The hemoglobin was 7.6 g/dl, and the hematocrit was 23.9%. The peripheral blood smear revealed a reticulocyte count of 5% (1% corrected) with marked basophilic stippling. Additional laboratory studies that were normal included serum iron, total iron binding capacity, serum protein electrophoresis, antinuclear antibody, direct and indirect Coombs' tests, and toxicology screen. The hemoglobin was type AA, and the erythrocyte sedimentation rate was 28 mm/h.

On the eighth hospital day, the patient was found unresponsive on the floor with her eyes tonically deviated to the right. Another probable seizure occurred on the ninth day, and administration of phenytoin and phenobarbital was begun. On neurologic examination, the patient had difficulty following instructions. The cerebrospinal fluid was sterile and contained 15 white blood cells (87% lymphocytes); the protein was 130 mg/dl, and glucose was 73 mg/dl. An electroencephalogram on the tenth day revealed diffuse slowing and the occurrence of epileptiform discharges over both frontal regions. Over the next three days her mental status gradually deteriorated, and she had additional seizures, despite adequate serum anticonvulsant levels. Computerized tomography with contrast enhancement showed increased uptake in a gyriform pattern throughout the brain (Fig. 1). The patient died on the 15th hospital day. A blood specimen obtained at autopsy and measured by atomic absorption spectroscopy contained 525 μg/dl of lead. The brain tissue showed massive swelling, with large amounts of proteinaceous material in the intercellular space (vasogenic edema).

Case 2. A 35-year-old black man sustained a gunshot wound to the abdomen requiring a nephrectomy 12 years prior to his present illness. The bullet remained in the L3–L4 disc space. In May 1980, he developed severe abdominal pain associated with nausea and vomiting of coffee ground material. A nasogastric aspirate was positive for occult blood, but the stool was negative. The hemoglobin was 10 gm/dl, the hematocrit 31%, and the MCV was 88 μ^3; the patient was released with the presumptive diagnosis of alcoholic gastritis.

From the Departments of Neurology and Surgery, University of Texas Health Science Center at Dallas, and the Department of Geology, University of Texas at Dallas, Dallas, Texas

* Department of Neurology.
† Department of Geology.
‡ Department of Surgery.
Reprint requests: Erwin R. Thal, M.D., Department of Surgery, University of Texas Health Science Center, 5323 Harry Hines Boulevard, Dallas, Texas 75235.

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TABLE 1. *Laboratory-Confirmed Cases of Lead Poisoning From Retained Missiles*

Case (Reference)	Clinical Features				Clinical Signs		
	Type of Projectile	Location	Time Intervals	Associated Disease	GI	Hm	Pn/En
1	Bullet	Femur*(?)	5 mos	Arthritis	+	+	−/+
2	Bullet	Lumbar spine†	12 yrs	Alcoholism, delirium tremens, thyrotoxicosis	+	+	+/−
3	Bullet	Chest wall† soft tissues	4 yrs	Alcoholism, fever	+	+	+/−
4 (4)	Bullet	Hip*	26 yrs	Arthritis, alcoholism, renal failure	+	+	−/+
5 (3)	Bullet	Knee*	3 yrs	Thyrotoxicosis, arthralgia	−	+	−/−
6 (24)	Bullet	Hip, arm, chest*	26 yrs	Arthritis, alcoholism, delirium tremens, acute renal failure	+	+	−/+
7 (25)	Bullet	Ankle*	40 yrs	Arthritis	+	+	−/−
8 (26)	Buckshot	Chest wall†	3 mos	Soft tissue abcess	+	+	+/+
9 (27)	Buckshot	Femur, thigh	3 days	Fracture, shock, fever	+	+	−/+
10 (28)	Buckshot	Chest, legs	2 yrs		+	−	−/−
11 (29)	Bullet	Metarcarpal joint	10 yrs	Arthritis	+	. . .	−/−
12 (30)	Bullet	Knee*	27 yrs	Arthritis	+	+	−/+
13 (31)	Bullet	Elbow*	26 yrs	Arthritis	+	+	+/−
14 (32)	Schrapnel	Head of humerus	10 yrs		+	+	−/+
15 (33)	Schrapnel	Pectoral muscle	10 yrs	HTN Atherosclerosis	−	+	+/−
16 (34)	Bullet	Head of femur	1 yr	Pyelonephritis	+	+	+/+

Case (Reference)	Laboratory Diagnosis, and Therapy			
	Urine Pb ug/L	Blood Pb ug/dl	Treatment	Outcome
1	206	525	None	Death
2	148–350	140	Propylthiouracil, EDTA & BAL, penicillamine, surgery × 2	Complete recovery over 1 yr
3	80	67	Penicillamine, surgery	Gradual recovery
4 (4)	. . .	353	EDTA & BAL, Total hip arthroplasty, penicillamine	Recovery over ? period
5 (3)	202	107	Propylthiouracil, EDTA & BAL, penicillamine, surgery	Recovery over ? period
6 (24)	. . .	350	EDTA & BAL, Total hip arthroplasty, penicillamine	Recovery over 3 mos
7 (25)	232–720	. . .	BAL & EDTA, surgery	Asymptomatic after medical regimen
8 (26)	400	. . .	Surgery, EDTA, surgery	Recovery over ? period
9 (27)	150	425	Surgery × 2, EDTA	Recovery over ? period
10 (28)	95	88	EDTA	. . .
11 (29)	300	60	Surgery	Recovery over 1 mo
12 (30)	190	. . .	Calcium gluconate + Sodium citrate	Death
13 (31)	. . .	60	Ca^{++}	Slight improvement over 1 mo
14 (32)	55–170	175–350	Surgery, sodium bicarbonate, surgery	. . .
15 (33)	110–220	40–50	None	. . .
16 (34)	0.2 gm / Many liters	. . .	Surgery	Died 3 days postop

Time Interval = injury to diagnosis; . . . = not available or done; * = within synovial fluid; † = within fluid filled cyst; GI = colic, constipation, nausea, vomiting; Hm = basophilic stippling, anemia; Pn = peripheral neuropathy; En = Encephalopathy.

In July, the patient returned because of recurrent abdominal pain and coffee ground vomitus. Orthostatic hypotension, tachycardia, a hemoglobin of 5 gm/dl, a hematocrit of 15%, and an MCV of 82 μ^3 were noted. Within hours of admission, he developed florid delirium tremens for which he received diazepam. The hematocrit stabilized at 40% after transfusion of 8 units of packed cells. On the fourth day, the patient had severe proximal weakness of all four extremities, with no involvement of bulbar or ocular musculature. No improvement was noted after administration of 10 mg of edrophonium intravenously. There was marked action tremor of the mouth, face, and hands. Although he was areflexic throughout with flexor plantar responses, his mental status and sensory examination were normal. A blue line at the gingival margin was noted. Additional laboratory tests were consistent with the diagnosis of hyperthyroidism, for which he was treated with propylthiouracil. The cerebrospinal fluid was acellular, with a protein of 120 mg/dl. The urine "heavy metal screen" was negative, and a determination of blood lead by atomic absorption spectroscopy was 41 μg/dl (intoxication > 60 μg/dl). The initial clinical impression was alcoholic myopathy or hyperthyroid myopathy. The vital capacity decreased from 2200 ml on day 4 to 460 ml on day 12, and the weakness progressed to total proximal paralysis and severe distal weakness requiring ventilatory support. A muscle biopsy on day 13 showed acute denervation, and the clinical diagnosis was changed to Guillain-Barré syndrome. Quantitative urinary porphyrins showed negligible amounts of porphobilinogen but significant excretion of coproporphyrins, 1.52 umole/l (N < 0.18 umole/1).

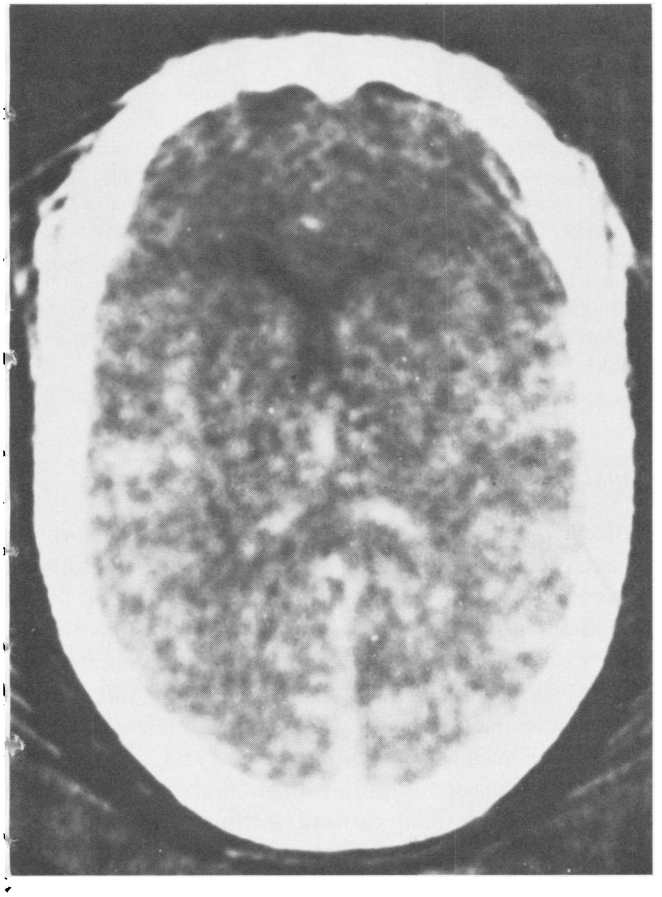

FIG. 1. Computerized tomography from patient 1 showing diffuse contrast enhancement.

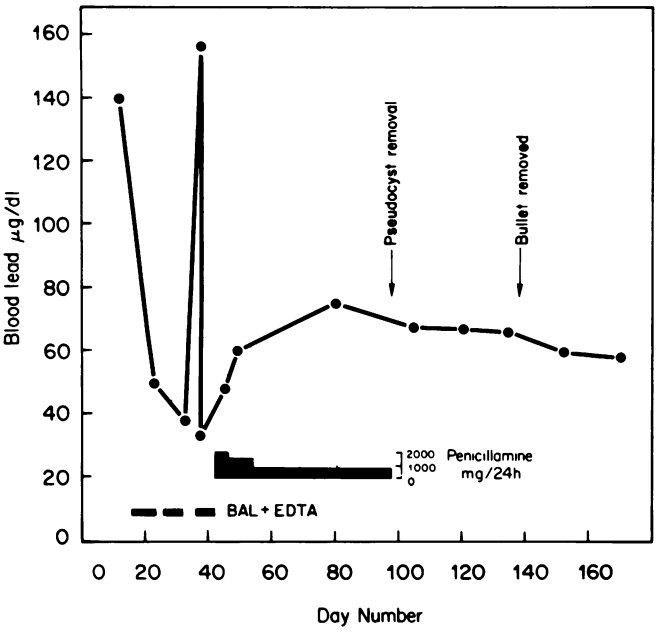

FIG. 2. Serial lead determinations from case 2. The concentrations were determined by stable isotope dilution mass spectroscopy. Three courses of BAL + EDTA chelation therapy were given. The bullet was removed on the 96th day.

A definitive diagnosis was made on day 14, when the blood lead determination was repeated by an alternate methodology (mass spectrometric stable isotope dilution) and was 140 μg/dl. The urine contained 405 μg Pb/24 h (N < 14 μg/dl). The patient was treated with three courses of chelation therapy using 150 mg of dimercaprol (BAL) IM every eight hours and 1.5 gm of calcium disodium edetate ($CaNa_2$ EDTA) IV every 12 hours as indicated. During chelation there was a large increase in the excretion of lead in the urine. Serial determinations of blood lead (determined by stable isotope dilution) during chelation are shown in Figure 2. Chelation therapy acutely lowered blood lead to less than the toxic level, with a rapid rebound after chelation was discontinued. The patient gained strength in the distal extremities, and his vital capacity increased to 800–1000 ml.

After the initial detoxication, it was decided to attempt to maintain the blood lead level at less than 80 μg/dl with D-penicillamine. He received penicillamine at a total daily dosage of 1500 mg for five days, then 1000 mg for ten days and 750 mg for 42 days. Pyridoxine was also administered to prevent pyridoxine deficiency neuropathy. During this period, urinary lead excretion remained between 2 and 3 mg/24 hrs, and the blood lead between 67 and 55 μg/dl. The patient's strength improved gradually, and he underwent abdominal surgery on day 96. A pseudocyst that communicated with the L3–L4 disc space was encountered in the prevertebral area and was removed. The tissue of the pseudocyst wall contained many lead fragments. The pseudocyst contained a thick grey fluid having the consistency of paint, with a large amount of suspended black material. After centrifugation, a large black precipitate that was x-ray dense was obtained (Fig. 3). The precentrifugation lead content of this fluid was 18.0 mg/gm and the supernatant was 6.3 mg/g. A trabecular bone specimen obtained from the iliac crest had a total lead content of 94 μg/gm. The bullet in the L3–L4 disc space was removed by a posterior approach on day 137, and the surrounding structures were curetted in an attempt to remove as much lead as possible. Following surgery, the blood lead gradually decreased without further chelation therapy (Fig. 2), and the patient's strength returned to normal.

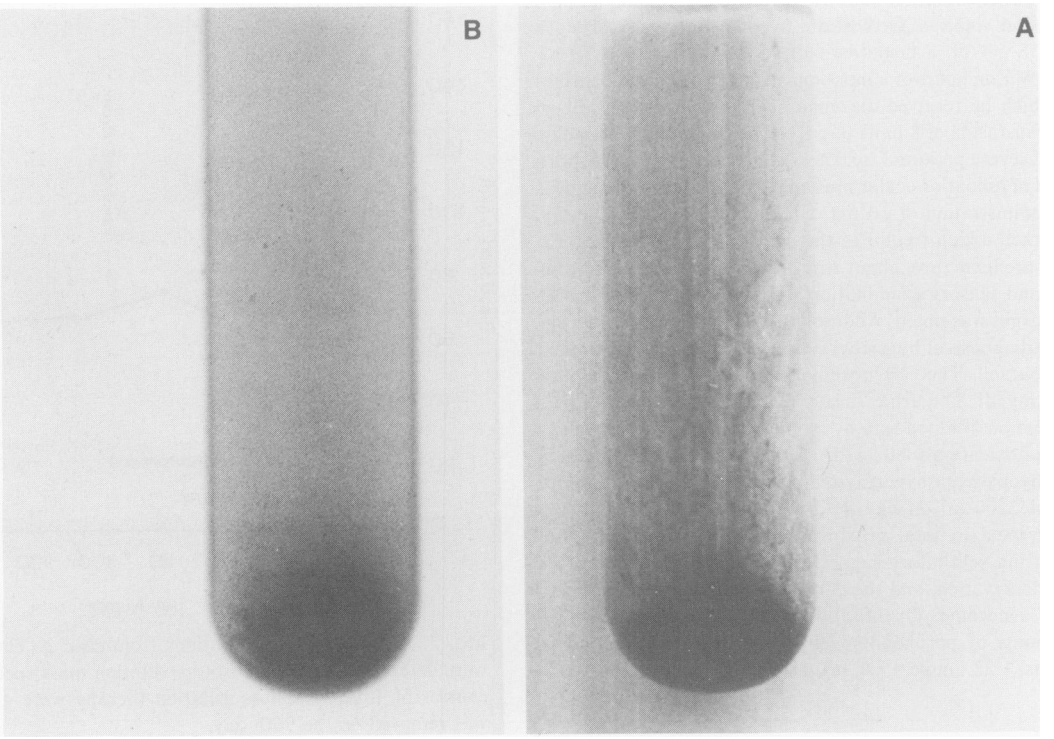

FIG. 3. Pseudocyst fluid obtained from case 2 and centrifuged showing a large black pellet (3B). An X-ray of the tube shown in Fig. 3B was obtained and is printed as a positive in Fig. 3A.

Case 3. A 27-year-old black woman presented with a six-month history of depression and a 40-pound weight loss. She had progressive generalized weakness, a 13-week history of severe dysaesthesias in the legs, poor fine motor movements in the hands, and increasing difficulty in walking, culminating in a three-day history of complete inability to rise from her bed. As summarized in Figure 4, she had been episodically ill with a variety of symptoms since her gunshot wound four years prior to admission. The gunshot wound had resulted in multiple retained bullet fragments in the right posterior chest wall.

On admission she was cachectic and chronically ill, with a fever of 38 C, and a pulse of 120. Her mental status and cranial nerves were normal. Palpation of her legs revealed extreme tenderness. Muscle strength was (MRC scale): deltoid 4/5, brachial radialis and biceps 3/5, wrist extensors 2/5, fingers 2/5, hamstrings 2/5, quadriceps 2/5, and dorsiflexors of the feet 1/5. Sensory examination showed markedly decreased vibration and position sense in the legs, and slight decrease in the arms. Light touch was decreased over the legs below midcalf. She exhibited hyperalgesia in the legs. Deep tendon reflexes were 2/4 in the arms and 0/4 in the legs. Plantar responses were flexor.

The laboratory examination revealed a hemoglobin of 10.6 gm/dl and a hematocrit of 31.4% with basophilic stippling. The serum folate, iron, and total iron binding capacity were normal. The initial blood lead was 67 μg/dl by atomic absorption spectroscopy. The urine lead was 80 μg/24 hrs/400 mg creatinine before, and 1104 μg/24 hrs/800 mg creatinine after EDTA stimulation. Motor nerve conduction velocity of the median nerve was 52 m/sec, with a distal latency of 4.0, and in the ulnar nerve 49 m/sec, with a distal latency of 2.8 m/sec. Electromyography of the left anterior tibial group showed evidence of denervation, with frequent positive sharp waves and fibrilations. Subsequent blood lead determination four days after EDTA challenge showed 54 μg/dl and 58 μg/dl using mass spectrometric stable isotope dilution (Fig. 4).

A shifting right subscapular bursa was noted which felt gritty. Radiologic examination confirmed the presence of multiple bullet fragments. The patient was started on penicillamine (Fig. 4). The 20 cc pseudocyst and bullet fragments were removed surgically. The pseudocyst contained a thick grey fluid with a finely suspended black material. This was centrifuged and revealed dense precipitate on x-ray. A cortical bone biopsy of the scapula contained 281 μg/gm of lead, while trabecular bone contained 483 μg/gm bone. Over the following year she improved from a Karnovsky scale[1] of 20% (moribund) to one of 70% (at home and able to care for herself, but unable to work).

Methods

Analytical Techniques

Lead levels were determined by atomic absorption spectroscopy at the Southwestern Institute of Forensic Sciences (Dallas) and by stable isotope dilution at the Department of Geology, University of Texas at Dallas. For atomic absorption spectroscopy, equal volumes (1.0 ml) of blood and concentrated nitric acid were mixed in screw-capped glass tubes. The specimen was heated in a water bath for 60 minutes at 85–95 C. The extract was removed for atomic absorption spectroscopy with carbon rods at the following settings: 110 C, 30 sec; Ash, 400 C, 35 sec; and atomize, 2400 C, 0 sec.

For the isotope dilution assay, 5 ml of blood was transferred to a 100 ml Teflon® bottle, dried and then decomposed in 20 ml of concentrated nitric acid by

slowly heating and then boiling until the evolution of brown fumes ceased. The process takes about 12 hours. An aliquot of the decomposed blood was spiked with 1 μg of isotopically pure ^{206}Pb. After stirring thoroughly to mix the spike and the endogenous lead, the combined lead was coprecipitated with barium nitrate. The lead was then separated from the barium by electrodeposition and isotopically analyzed on a thermal ionization, 12-inch radius, single focusing, 60° sector field mass spectrometer. This method of assay is essentially the same as that used at the National Bureau of Standards.[2]

Special Studies

The concentration of lead was measured in an iliac bone biopsy specimen obtained at the first operation in case 2 and in a portion of the scapula of case 3 containing the embedded projectile. The specimen was taken approximately 5 mm from the projectile itself and contained no lead fragments. The bone specimens were cut into slices about 0.5 mm thick and boiled in chloroform. The cell debris were scraped from the bone surfaces with a needle, and loosely adhering particles were removed by ultrasonication in distilled water. The specimen obtained from case 2 was entirely of trabecular bone. Cortical and trabecular bone were analyzed separately in the specimen from case 3. All lead assays were performed by the stable isotope dilution method. The fluid from the pseudocyst in case 2 was studied so as to determine the distribution and chemical nature of lead in this material. The fluid contained 18 mg Pb/g. After centrifuging at 10,000 g × 20 min, the supernatant contained 6.3 mg Pb/g, and a pellet of black material was obtained. This pellet was freeze-dried, then sonicated in bromoform (S.G. → 2.89). After agitation, the suspension was transferred to a separatory funnel, and the dense particles allowed to settle. This material was examined by scanning electron microscopy, electron probe microanalysis, and x-ray diffraction.

Discussion

Clinical Diagnosis

The protean manifestations of lead poisoning, which include anemia, abdominal colic, nephropathy, encephalopathy, and motor neuropathy, are well-known. Recognition of these symptoms as manifestations of lead poisoning in the patient with a retained missile is made more difficult by the intermittent nature of the symptoms in some cases and by the presence of an intercurrent illness in other cases. Scattered reports, summarized by Cagin[3] and Dillman,[4] indicate that symptomatic individuals with chronic lead poisoning develop clinical plumbism during periods of metabolic

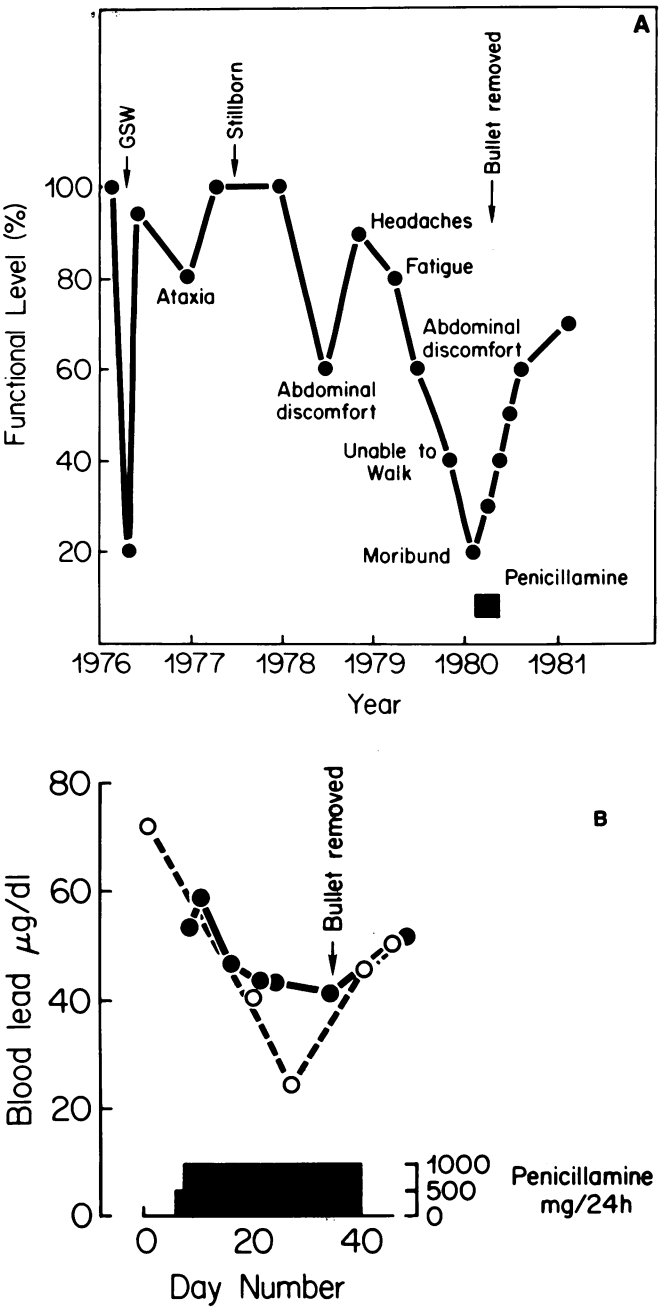

FIG. 4. A. Clinical symptomatology during four years of patient 3 following a gunshot wound to the chest. She developed intermittent symptoms including a stillbirth, abdominal crises, and progressive weakness. Following removal of the bullet, she made a gradual recovery over a period of one year. Ratings were made using the Karnofsy scale. B. Lead levels measured by mass spectroscopy (solid line) were greater in the lower ranges than those measured by atomic absorption spectroscopy (broken line).

change affecting bone (*e.g.*, acidosis, hyperparathyroidism) or the nervous system (viral meningitis), or both (hyperthyroidism, acute infection). Although still controversial, alcohol has also been claimed to precipitate the symptoms of lead poisoning in patients with

chronic exposure to lead.[5,6] With mobilization of the stored lead in bone, the soft tissue and blood levels increase, and clinical symptoms occur. The associated diseases in the previous 13 cases are shown in Table 1. In this regard our case 1 had an underlying arthritic condition, which limited her physical activity which may have contributed to increased mobilization of bone. In case 2 possible precipitating factors included alcoholism, delirium tremens, and thyrotoxicosis. This is the second reported case of plumbism from a bullet associated with concurrent transient thyrotoxicosis (case 5). Our case 3 had severe alcoholism, fever, and inactivity as possible precipitating factors. In this case, it is important to emphasize that this patient had an underlying alcoholic sensory neuropathy in combination with a severe motor neuropathy out of proportion to the usual motor changes in alcoholic neuropathy. Her peripheral nervous system disease was undoubtedly caused by the combination of both alcoholism and plumbism. The latter has always been associated with motor neuropathy and spares the sensory fibers. It is also possible that the damaged sensory fibers were more vulnerable to subsequent exposure to lead.

Radiologic examinations can be helpful in supporting a diagnosis of plumbism. Recognition of a retained missile or fragments can document a potential source of lead intoxication, especially if lodged in bone or near a joint space or associated with a pseudocyst. The computerized tomography scan of the head in case 1 correlated well with the pathologic findings at autopsy. This is the second reported case of computerized tomography of the brain in lead encephalopathy.[7] Both cases demonstrate contrast enhancement in a gyriform pattern.

Laboratory Diagnosis

The problem of diagnosing plumbism from retained bullets stems not only from a lack of clinical awareness, but also from the laboratory methods used for confirmation. The laboratory confirmation of lead poisoning is neither straightforward nor easy. The "heavy metal screen" that is a widely employed qualitative test on urine was negative in all three of our cases; we have not encountered a patient with lead, arsenic, mercury, or thallium poisoning in whom this test has been positive among a total of nine confirmed cases of heavy metal poisoning in the past year. More specific tests for lead in the blood and urine are available but must be interpreted with great caution. In clinical laboratories, the most widely employed technique for determining lead uses atomic absorption spectroscopy, although several reports document that this measurement can be unreliable.[8-14] Proficiency testing in 1975 among different laboratories using these methods revealed that in over 50% of the laboratories, the results were invalid.[8,9] Measurements by atomic absorption spectroscopy are inaccurate whenever the biological sample contains a substance that suppresses the emission, a situation that can be detected only if a second determination is made after a measured quantity of the lead has been added to the specimen.[15] This type of internal standardization is rarely performed. A recent comparison of results of lead determinations in clinical laboratories and of the definitive method using isotope dilution mass spectroscopy showed that the two methods tended to agree well in the restricted region around 40 μg/dl.[11] We did not attempt to make a point-by-point comparison of the two methods, but the data in the literature[8,9,11,12,14,15] would indicate that the initial incorrect lead level in case 2 was not a spurious result in an isolated laboratory.

Alternative methods for the confirmation of lead poisoning that do not involve the direct determination of blood or urine lead are available. These methods rely on the fact that lead inhibits several enzymes involved in heme biosynthesis, including ALA synthetase, ALA dehydrase, and coproporphyrinogen decarboxylase. The degree of inhibition of each of these enzymes depends on the blood lead level, the duration of exposure, and the effect of feedback mechanisms on enzyme levels. ALA dehydrase has an exact linear relationship with blood lead concentration, whereas urinary ALA and coproporphyrin excretion begin to rise when blood lead exceeds 35–40 μg/dl.[16,17] The relationship between the excretion of ALA and blood lead concentration is curvilinear.[16,17] Measurement of free erythrocyte protoporphyrin is a recently devised test that has an exponential relationship with blood lead levels.[18] The increased excretion of ALA and coproporphyrin was an important clue leading to the diagnosis in our second case.

Elevated blood lead, even if the measurement is accurate, does not prove lead intoxication but shows, instead, recent exposure or absorption of lead. Although the finding of elevated blood lead (or a corresponding change in one of the enzymes of porphyrin metabolism) in the presence of characteristic clinical symptomatology is highly significant, blood and urine lead concentrations can correlate poorly with total body stores and symptoms in chronic lead poisoning.[6,19] For patients with retained missiles who are having intermittent symptoms (*e.g.*, colic) that might be caused by lead toxicity, lead mobilization by calcium disodium edetate ($CaNa_2EDTA$) infusion can be a useful diagnostic test. Recently, the fluorescence of zinc protoporphyrin in erythrocytes has been shown to be a sensitive indicator of body stores of lead and may well become useful in following or detecting patients with plumbism.[20,21]

FIG. 5. Scanning electron micrograph of isolated particles from pseudocyst fluid from case 2.

Pathogenesis

Metallic lead is insoluble, and most patients with retained lead missiles are under no threat of systemic absorption of the lead. The cases presented illustrate that under certain conditions the lead from a retained missile can be redistributed. In two of the cases, the bullet was associated with a pseudocyst, which was undoubtedly involved in the redistribution of the lead from the missile. The exact sequence of events in the mobilization of the lead from the bullet is still unclear. The wall of the pseudocyst contained radiopaque deposits, which were presumably some form of lead. The fluid within the pseudocyst contained large amounts of lead (18 mg/gm in case 2), which could be subfractionated by centrifugation into a soluble form (6.3 mg/gm), and an insoluble, black, finely divided radiopaque material (Fig. 3). This material was isolated; in the scanning electron microscope, aggregates of irregularly shaped particles 5–50μ in the largest dimension were noted (Fig. 5). These particles were examined with the electron microprobe, and the presence of lead was confirmed. Based on the appearance and color of this material, PbS was suspected, but no sulfur was detected. The instrument is incapable of identifying elements of atomic number 8 (oxygen) or less. The particles were identified as lead dioxide using x-ray diffraction. It is not clear whether the various forms of lead found in the pseudocyst indicate that the lead in the bullet is first removed in a soluble form and then precipitated and/or if the surface of the bullet is oxidized to the very friable compound lead dioxide.

The removal of the lead from the pseudocyst may involve several mechanisms. The soluble lead may diffuse into the general circulation. The insoluble lead could be removed from the pseudocyst by phagocytosis. This mechanism is analogous to events known to occur after exposure to lead dioxide (PbO_2) by workers who handle metallic lead.[5] Lead dioxide is a highly friable material that forms on the surface of metallic lead and is inhaled as a dust, which is then removed from the respiratory tract by macrophages.

The location of the missile in the body may also be important for redistribution of the lead. In seven of the 13 previously reported cases, part or all of the missile was in contact with joint fluid. Five of these seven cases had an associated arthritis, and one had arthralgia, which was probably secondary to the irritation within the joint space. It has been assumed that some factor in the synovial fluid leads to accelerated solution of the

lead, but this is unproven. The concentration of lead and identification of the lead compounds in the joint fluid have not been reported. The associated arthritis with the concomitant increased vascularity and inflammatory cells may well be a sufficient explanation for the systemic absorption of lead from a joint space. The disc space (case 2) is atypical in that it is highly avascular and, therefore, is unlikely to have been the site of absorption, compared with the adjacent pseudocyst. In case 3, the subscapular bursa contained a pseudocyst, which in turn contained the lead. These observations would suggest that bullet fragments outside of synovial cavities can also be a potential source of lead toxicity, especially in the presence of a pseudocyst.

Treatment

An established principle of toxicology is the removal of the patient from the toxic source as the first avenue of therapy. When the toxic material is a shattered bullet within the patient, removal of the toxic material may present special problems. Surgery to remove the lead fragments is not usually indicated as the *initial* therapy for the patient with documented plumbism from retained missiles. The patient should be stabilized and the lead burden reduced by chelation therapy. Once the patient has been stabilized, the administration of D-penicillamine at a dosage that keeps the blood lead below 80 μg/dl will allow the patient to improve to the point at which surgery can be safely performed to remove the tissues containing the lead. The chief indication for operative intervention is the prevention of future episodes of plumbism.

Three of the cases presented in Table 1 became severely symptomatic following surgery without prior chelation therapy (cases 8, 9, 16). In fact, the patient in case 16 died of acute lead encephalopathy postoperatively. This suggests that surgery may mobilize bone lead stores and result in acute plumbism. For example, in case 3, the lead content of the trabecular bone was 483 μg/g, and that of the cortical was 281 μg/g. In case 2, even after prolonged chelation therapy, the lead content of trabecular bone from the iliac crest was 94 μg/g. A normal value for lead in cortical bone of 15 μg/g and in trabecular bone of 8.6 μg/g had previously been measured in a specimen taken from one of the authors (WIM), whose blood lead concentration was 14 μg/dl. Multiple factors including bone or tissue manipulation, stress, physical inactivity, or metabolic imbalances may be involved. These hazards suggest that chelation therapy prior to surgery is indicated for all patients with plumbism from retained missiles.

Of special concern during chelation therapy is the theoretical possiblility that these drugs will aggravate the plumbism. Our cases 2 and 3 and others in the literature (4, 5, 6, 7) document that chelation therapy in the presence of a retained missile is both safe and efficacious for the treatment of symptomatic plumbism. However, both the medical and surgical treatment of this unique form of plumbism are potentially hazardous in other ways. Surgical removal of the tissues containing the lead should be performed with the awareness that much of the lead may be in the form of a liquid suspension. If this toxic material is spilled, it must be removed by copious irrigation. The patient should be followed closely after surgery for reoccurrence of acute plumbism.

Prevention

The number of patients at risk for plumbism from retained missiles may be surprisingly large, yet few cases have been recognized. Until the true incidence of this complication is known, it is not recommended that patients with gunshot wounds be subjected to extensive surgery to remove the lead fragments. However, patients with retained missiles should be considered at risk for lead poisoning and followed carefully. Lead fragments in a joint space have a higher potential risk, and surgery to remove the lead fragments in the joint space may be indicated to prevent both a localized traumatic arthritis and plumbism.

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